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CORRESPONDENCE

Psoriasis herpeticum: Kaposi's varicelliform eruption in psoriasis



Dear Editor,

A 45-year-old man had a past history of psoriasis and pulmonary tuberculosis with postantituberculosis treatment status. Psoriasis was diagnosed from typical clinical presentation and histopathology findings. He received irregular treatment with oral methotrexate and topical steroids for psoriasis. The patient was admitted for inpatient treatment because of severe sepsis and erythrodermic psoriasis (Figure 1). However, he developed multiple severe painful ulcerations on the abdomen, back, and buttocks for 3 days. Some preceding itchy erythematous papules were noted on the lower abdomen 2 days before this episode. No obvious herpes labialis, herpes genitalis, or herpes zoster infection were found.

On physical examination, he had widespread and progressive clusters of painful punched-out ulcerations on the abdomen, back, and buttocks (Figure 2A and C). Tzanck smear from the bases of these lesions tested positive for multinucleated giant cells and keratinocytes with ballooning change (Figure 2B). We also performed a skin biopsy of the erythroderma to rule out other possibilities; the histopathological diagnosis was psoriasis. In addition, multiple hyperkeratotic papuloplaques and excoriations were noted on the interdigital folds of the hands and bilateral

axillae. Skin scrapings from these lesions by potassium hydroxide examination revealed the presence of scabies mites, and the lesions were diagnosed as scabies infestation. The patient was started on a regimen of parenteral acyclovir sodium (Zovirax 250 mg, 3 times daily for 14 days), parenteral teicoplanin (Targocid 400 mg daily for 14 days) for methicillin-resistant *Staphylococcus aureus* bacterial infection, and oral ivermectin (Stromectol 12 mg once and repeated at Day 14) for scabies infestation. Topical silver sulfadiazine cream twice a day was used for wound care. There were significant re-epithelialization and healing after the treatment.

Kaposi's varicelliform eruption (KVE), first described by Moritz Kaposi in 1887, is a widespread cutaneous infection with herpesvirus type 1 or type 2, vaccinia virus, or coxsackievirus A16 in a patient with underlying dermatoses.¹ The term “eczema herpeticum” is used if herpesvirus type 1 or type 2 is the causative pathogen. KVE is most often seen in patients with underlying atopic dermatitis. In addition, multiple underlying dermatoses associated with KVE have been reported, including pemphigus foliaceus, Darier disease, pemphigus vulgaris, pityriasis rubra pilaris, Hailey-Hailey disease, irritant contact dermatitis, cutaneous T-cell lymphoma, seborrheic dermatitis, Wiskott-Aldrich syndrome, congenital ichthyosiform erythroderma, and Sezary syndrome.^{2–4} However, KVE rarely occurs in psoriasis and only a few cases have been reviewed in literature. In 2002, Saraswat et al first described KVE in a patient with erythrodermic psoriasis.⁵ The term “psoriasis herpeticum” was coined by Santmyire-Rosenberger et al to describe the occurrence of KVE in psoriasis.⁶

KVE may begin as painful vesiculopustular, sometimes umbilicated, lesions which then progress to punched-out ulcerations in the area affected by the underlying dermatoses. The lesions most commonly appear on the face and often spread in 7–10 days.⁷ The lesions may coalesce into large, ulcerated, painful plaques that can be easily superinfected with bacteria and yeast.⁸ KVE can be associated with fever, malaise, and lymphadenopathy. The average duration of KVE has been estimated to be 16 days, and lesions most often heal without scarring in 2–6 weeks.⁸

Risk factors for KVE reported in literature include erythroderma, systemic sepsis, therapy with immunosuppressants such as methotrexate and systemic steroids, and therapy with systemic retinoids.⁶ In a case series, erythroderma was noted in 35% of patients with KVE. The underlying dermatoses for KVE with erythroderma included airborne contact dermatitis (15%), psoriasis (15%), and cutaneous T-cell lymphoma (5%).⁹ In the present case, the patient concurrently suffered from a scabies infestation. However, scabies infestation associated severe itching, excoriation, and broken skin

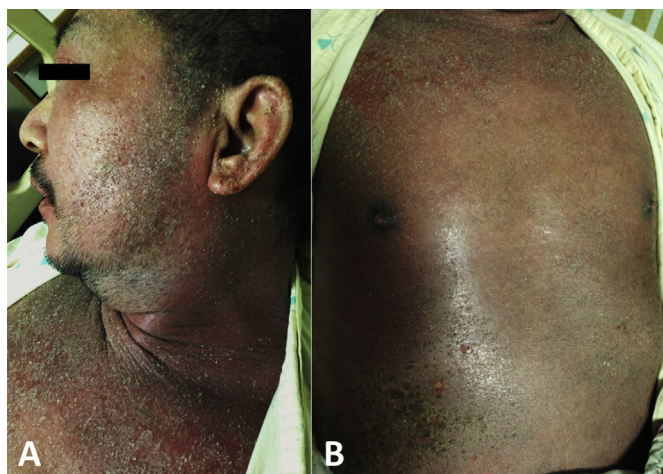


Figure 1 Generalized scaly erythematous patches and plaques on the face, trunk, and limbs.

Conflicts of interest: The authors declare that they have no financial or non-financial conflicts of interest related to the subject matter or materials discussed in this article.

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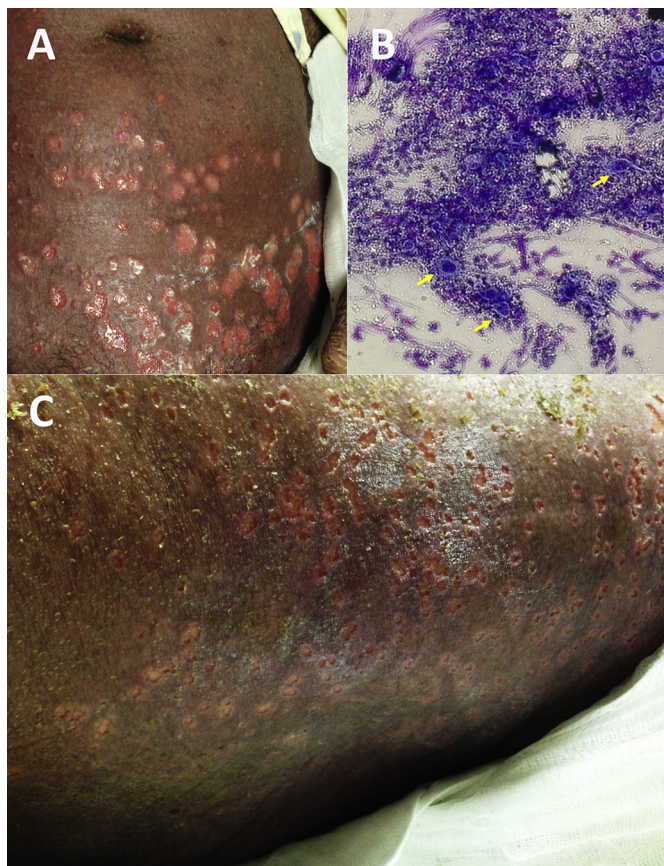


Figure 2 (A) (C) Widespread and progressive clusters of painful punched-out ulcerations on the abdomen, back, and buttocks; (B) Tzanck smears of these lesions were positive for multinucleated giant cells and keratinocyte with ballooning change (yellow arrows). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

barrier may increase susceptibility to KVE. We thus suspect that concurrent scabies infestation may also be a risk factor for KVE.

The differential diagnosis of KVE includes impetigo, irritant contact dermatitis, and chemical corrosive injury. Physical examination, laboratory testing, and careful recording of the patient's history will aid diagnosis. A Tzanck smear, microscopically showing the presence of multinucleated giant cells and keratinocytes with ballooning change, can confirm the presence of a viral infection. Direct immunofluorescence which provides more sensitive and specific results using unique herpes virus antibodies can be performed in a specialized laboratory. Viral cultures can also help to confirm the diagnosis of KVE, but it takes several days.⁴

Immediate antiviral therapy is essential in patients with KVE. Intravenous acyclovir (10–15 mg/kg/day) and oral acyclovir

were deemed effective.⁷ If superinfection with bacteria and yeast occur, treatment with antibiotics and an antifungal agent is necessary.

Psoriasis herpeticum due to varicella zoster virus (VZV) has been reported recently.¹⁰ KVE with prominent herpetiform segmental vesiculopustular eruptions favor the diagnosis of VZV-related psoriasis herpeticum. Comparing our case with that describing VZV-related psoriasis herpeticum,¹⁰ both reports describe patients who were febrile, erythrodermic, and under immunosuppressive therapy. However, we found neither zosteriform distribution nor disseminated lesions in our patient. Both cases responded well to acyclovir therapy.

Here, we reported a rare case of psoriasis herpeticum, KVE, in a patient with erythrodermic psoriasis and severe sepsis. Early recognition of KVE in patients with psoriasis, identification of the associated risk factors, and early initiation of antiviral therapy will lead to a better prognosis in such patients.

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